PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, January 23, 1893.

The President, Dr. FRANCIS X. DERCUM, in the chair.

Dr. H. F. HANSELL, read a paper entitled

THE POSSIBILITY OF THE EARLY DIAGNOSIS OF LOCOMOTOR ATAXIA FROM THE EYE-SYMPTOMS.

The eye symptoms of the earliest stage of tabes may be described under three heads, those arising from affections of the extrinsic muscles, of the pupil and of the optic nerve. A number of observations from clinical cases and pathological studies has been recorded to prove that partial or complete paralysis of any one of the straight or oblique muscles or the elevators of the lids of one or both eyes, is at times a fore runner of ataxia. The muscles most frequently affected are the external rectus and the levator palpebrarum giving rise to ptosis and crossed diplopia. A strong diagnostic feature between paralysis from tabes and that from other causes, is that in the former the paralysis is incomplete and transitory. In the late stages when the nuclei in the floor of the fourth ventricle become implicated in the sclerotic process the paralysis recurs and the condition known as ophthralmoplegia results. The cause of the transient paralyses is ascribed by a few writers to nuclear disease, but the majority incline to the view of a vaso-motor disturbance.

The pupils are studied with reference to their size and shape and their reactions. Interference with the conduction of the sense of light, with its preception by the vasal ganglia, with the nuclei in the floor of the fourth ventricle or their connecting fibres, with the cilia-spinal centre in the cord, or with the pupillary branch of the third nerve, or vaso-motor fibres of the sympathetic, will determine anomalies of the pupil. Myosis is a characteristic of the tabetic pupil and must be regarded as a vaso-motor paralysis originating in disease of the centre. Berger in an analysis of 109 cases of tabes, followed through the three stages, declared that in all, the pupils were altered in size, and that this alteration was found in the great majority of cases, in the preataxic stage. The same writer and many others, have shown that the Argylle-Robertson pupil, not only is common in tabes, but is one of its earliest signs. A peculiarity of the contracted pupil of tabes is its medium dilatation to myonatics and the comparatively long time necessary for its return to its previous size.

Both myosis and the Argylle-Robertson pupil may precede altered locomotion and sensation by many years. The pupils may, also, be irregular in outline and unequally contracted.

The accomodation is seldom affected in the early stage. Two of the three cases briefly mentioned below have myosis and Argylle-Robertson pupils with no other spinal symptoms and showed other signs of tabes develop. They will furnish strong evidence in favor of the possibility ofdiagnosing that disease in its earliest stage, from the eye symptoms. The third case died from tabes eight years after myosis was first noticed.

The first sign of altered function of the optic nerve is limitation of the color field without narrowing of the field for white, deterioration of central acuteness of vision for form or opthalmoscopic changes. Later the field for white becomes concentrically limited, central vision declines, and the opthalmoscope shows the well known characteristics of non-inflammatory, incipient or advanced, atrophy. Emphasis should be laid on the superiority of the perimeter over the opthalmoscope in the early diagnosis of tabes. Dilatation of a retinal vein, which later returns to its normal calibre has been observed before evident involvement of the optic nerve. There is no doubt that incipient or even pronounced atrophy of the optic nerves may be a forerunner of tabes.[1]

CASE I. Mr. B., age 61.—Neurotic history. Pupils very small and have been so for ten years. L. slightly irregular in outline. Both respond to convergence but not to light. Four months ago he commenced to see double from a paresis of the L. external rectus. The double images were fused at my first examination by a prism of 12°, later by 30°, and, at my last examination, made two weeks ago, by 4°. The lower, nasal retinal vein was dilated, but soon regained its normal calibre. The color fields were temporarily limited, optic discs normal in color and outline. No general symptoms.

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[1] The presence of inflammatory exudation from the iris must be excluded and the repeated observations necessary to estimation of size be conducted with the same degree of illumination.)

CASE II. Mrs. V., age 55. Unilaterial myosis and Argyll-Robertson pupil. Pain in lower extremities, no alteration of fields, no paralysis or incoordination.

CASE III. Mrs. G., age 60. Myosis, loss of color perception and night blindness preceding for eighteen months, spinal optic nerve atrophy. Death eight years after first examination, from tabes.

Conclusions:

1. Transient ptosis or external ocular paralysis may, be one of the earliest symptoms of locomotor ataxia.
2. The Argyll-Robertson pupil, inocular or binocular, may precede for a variable length of time the general symptoms of locomotor ataxia.
3. Idiopathic, non-inflammatory optic nerve atrophy may be the first manifestation of locomotor ataxia.
4. Transient ptosis or diplopia, Argyll-Robertson pupil, incipient optic nerve atrophy associated in an individual past middle life, of inherited neurotic tendency, are strong presumptive evidence of the first stage of locomotor ataxia.

DISCUSSION.

Dr. H. C. WOOD.—I should like to call attention to one point which I think has not been dwelt upon and that is that the Argyll-Robertson pupil is not rarely seen reversed; in other words, there is preservation of the ocular reflex for light and loss of the reflex for accommodation. I think that this is not so common as the true Argyll-Robertson pupil, but it frequently occurs and has not been much spoken of. It is probably as significant as is the Argyll-Robertson pupil of organic disease of the nervous system.

Dr. JAMES HENDRIE LLOYD.—Cycloplegia occurs in some forms of multiple neuritis which simulate tabes. It is also observed in post-diphtheritic paralysis. I do not think it is common, as Dr. Wood states, to have the reversal of the Argyll-Robertson phenomena in true tabes. I have, however, seen cycloplegia come on in one or two cases of multiple neuritis, post-diphtheritic, simulating tabes in some of the symptoms. This might be called, on hasty observation, a reversal of the Argyll-Robertson pupil, although of course it is not truly so.

I was struck by a reference in Dr. Hansell's interesting paper to a form of ophthalmoplegia, I think external, coming on with symptoms of migraine, in the early stage of locomotor ataxia. Charcot, in a clinical lecture, has reported a case of that rare form of disease, "migraine ophthalmoplegique." It occurs to me that there might be some connection between this migraine and late coming tabes, especially as it is claimed by some that migraine is not an altogether uncommon predecessor of tabes or even of general paresis.

Dr. WHARTON SINKLER.—My experience has been the same as that of Dr. Lloyd as regards the reversal of the phenomena of the Argyll-Robertson pupil. I have seen two or three instances of it in tabes, but I think that it is not often met with.

Dr. Hansell's paper has interested me greatly. I really think that when we meet with cases of transient diplopia or ptosis, we ought to suspect the on-coming of tabes. I saw only a couple of days ago a patient who is in an instance of the truth of what Dr. Hansell has said. The gentleman who even now has only incipient tabes, has myosis, absence of knee-jerk and slight incoordination, but he has no ataxic pains. He has, however, well-marked disturbances of sensation with loss of thermal sense. On inquiry, he told me that several years ago, he had attacks of diplopia which were attributed to indigestion. This was six or seven years ago, before the other symptoms of tabes were observed.

Dr. J. MADISON TAYLOR.—Some time ago, I showed to the Society, a boy with Freidreich's ataxia, in whose case, the pupillary symptoms were reversed. I do not know whether or not this has any significance in that form of ataxia. In looking over the literature, I find the pupillary symptoms of Freidreich's ataxia are rather irregularly stated. In this case they were absolutely reversed.

Dr. CHARLES K. MILLS.—Dr. Hansell has given an excellent resume of these pre-ataxic eye symptoms, and the only remark that I have to make is that perhaps too much stress might be laid on these ocular symptoms in making the diagnosis of future ataxia. This might be misleading. It is true that these symptoms do frequently precede this disease, but on the other hand, it is true that in a fair percentage of cases, one or more or all of the ocular symptoms which have been mentioned, may be the precursor of some other form of degenerative disease, or indeed so far as the symptomatology is concerned may be the only clinical picture of the degenerative disease itself. While of course it is valuable to emphasize the importance of these symptoms, a word of caution should I think be given with reference to hastily making the diagnosis of coming ataxia from their presence. Myosis is seen in paretic dementia. It also occurs in pseudoparetic dementia, of syphilitic origin, and also in some forms of multiple neuritis.

Dr. H. C. WOOD.—I did not mean to be understood as saying that the reversed Argyll-Robertson pupil, but only that it was not very rare.

I recently reported in a clinical lecture to be published in *La Semains Medical* a case in which the diagnosis rested partly upon the pupillary condition. The case, I believe, is one of a new form of locomotor ataxia crisis which I have proposed the name of ataxic lympho-pathy. The patient is a man with a long history of exposure to the elements as a boat captain. When 58 years of age, he was seized with a frightful pain in the left angle of the jaw. This was associated with great swelling. He consulted his family physician, who believed that there was going to be suppuration of the gland. The pain amounted to agony. In three or four days, however, the swelling subsided without leaving any trace. Shortly afterwards the man had a similar attack and this has been followed by repeated attacks with frightful pain, with swelling of the glands, this swelling subsiding in a few days and the gland apparently going back to its normal condition. as a result of a series of the attacks, however, the cervical glands have become slightly enlarged.

Without going into the details of the case, I found that the character of the pain and swelling indicated a neurotic origin and this led to the suggestion that possibly I had a form of ataxic crisis to deal with. I found myosis with reversed Argyll-Robertson pupil, complete loss of the knee-jerks and marked retardation of the sensation in the legs with pronounced parasthesia, and that during the last three years, the man had gradually lost the power of equilibriation. I do not see how this case can be looked upon as anything else but a case of locomotor ataxia with crises affecting the region of the lymphatic glands, with enlargment of these glands.

The PRESIDENT.—I should like to remark that the history of swelling and painful crises related by Dr. Wood is somewhat like that of the cases of Adiposis

Dolorosa, which I have reported, in which was swelling of the tissues and in which shooting and darting pains accompanied the pain. Afterward the swelling would somewhat, though not altogether, subside. It is possible that Dr. Wood's case might be explained on the theory of neuritis.

Dr. H. C. WOOD.—In the case to which I have referred, there was no tenderness over the nerve trunks and there was evident enlargment of the lymphatic glands. Neuritis would hardly explain the loss in the knee-jerks, the retardation of sensation and the condition of the pupils.

Dr. H. F. HANSELL.—With the reversion of the Argyll-Robertson phenomenon, I should look for a different pathology. The paralysis of the sympathetic fibres to the pupil from destruction of or pressure upon the cilio-spinal centre, is accepted I believe, as the cause of the Argyll-Robertson pupil. If the condition is reversed, we should have to explain it by disease of the nucleus of the iris in the floor of the fourth ventricle.

Dr. Lloyd's statement that paralysis of the accomodation is found in central diseases of the nervous system is true, but that fact does not bear upon the eye symptoms found in the earliest stages of the ataxia since the accomodation is seldom affected in that disease. The pupil, which has its sympathetic centre in the higher part of the spinal cord, is the only one of the muscles within the eye, commonly involved.

In conclusion, I wish simply to say that my paper was designated "the possibility," and that its conclusions are not absolute because a large material is hard to collect and the cases difficult to follow to their termination, for reasons that will readily occur to you. The association of the three symptoms, however, transient paralysis of the external muscles, Argyll-Robertson pupil and loss of green in the visual field, would warrant a careful study, and search for other symptoms of tabes.